How do marine mammals survive deep diving?[1]

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INTRODUCTION

How long can you hold your breath? Most of us would be struggling to hold our breath for 60 seconds at rest. If, in addition to this feat, we had to swim to depth and then swim back to the surface without face mask or flippers, how deep could we go? Even fit human breath-hold divers would be struggling to reach a depth of only 10 m under these conditions (Kooyman 1988).

Eskimos, who hunted seals and whales from small boats, would have noticed the remarkable ability of these marine mammals to hold their breath under water for durations much longer than a human being. More recently whalers in sailing ships hunted a variety of species including the giant Sperm Whales. From the amount of cable run out to harpoons imbedded in these whales, they suspected that the depth of dives might be as great as 2 km.

One of the most intriguing questions then about marine mammals, is "How do they survive deep diving?"

The range of size of marine mammals is huge (Table 1). The Blue Whale is the largest animal known to have lived on earth, whereas the Bottle-nose Dolphin is only one-tenth the length and one 600th the weight of this leviathan.

Until very recently the Sperm Whale was believed to be the deepest diver (Table 2), but it has now been clearly documented that Elephant Seals are capable of dives greater than 1 000 m (Hindell, Slip and Burton 1989). A Northern Elephant Seal dived to 1 529 m, now the greatest depth recorded for an air-breathing vertebrate (De Long and Stewart 1989). Surprisingly, the Blue Whale has been documented to dive to only 100 m. This shallow diving may be related to its diet of small crustacea or krill which gather in vast swarms often near the surface. However, when stressed by harpooning for example, they may go to much greater depths (Hempleman and Lockwood 1978).

As for the time that these animals can spend under water on a single breath of air, the Sperm Whale and Weddell Seal can both tolerate a duration of over one hour (Table 2). Clearly, size *per se* is not an absolute indicator of resistance to underwater asphyxia.

The marine mammal which has probably been most investigated for its diving capacity is the Weddell Seal (Fig. 1) which is a native animal of Antarctica. Much of the information we have about the diving behaviour of the Weddell Seal comes from the work of Dr Gerald Kooyman of the Scripps Institution of Oceanography, San Diego, California. Dr Kooyman spent considerable time in the 1960s studying the diving behaviour of Weddell Seals at McMurdo Sound in Antarctica (Kooyman 1969).

For these studies a hut was set up on the sea ice at a location remote from any cracks or seal holes. An artificial hole was cut through the ice inside the hut. Seals were captured several kilometres away and brought to the hut where a small pack of instruments including a depth-time recorder was attached to the animal. The seals were then released through the hole into the water. With this simple technique Kooyman was able to determine both the depth and duration of dives.

When a seal is first released into the water it performs several *short exploratory dives.* Usually it will then embark upon a more *extensive exploratory dive* lasting from 20 minutes to an hour or more. These dives are generally no deeper than 100 m. A further type of dive is the *feeding dive* which can extend to a depth of 400 m and occasionally as much as 600 m but the duration is only 15 minutes or so. Incredibly, a series of feeding dives may continue for several hours!

What is Meant by Pressure?

At sea level the atmosphere, which extends to about 150 km above us, exerts a pressure on our bodies defined as one atmosphere absolute (ATA). One ATA is equivalent to the pressure exerted at the bottom of a column of mercury (Hg) 60 mm high. A depth of 10 m

Table 1. Length and weight of four of the better known species of marine mammal. The species are arranged in order of magnitude. The human is shown for comparison.

Species	Length m	Weight kg
Blue Whale (max.)	30	136 000
Sperm Whale (max.)	18	61 000
Weddell Seal	3	400
Bottle-nose Dolphin	3	200
Human	2	70

of sea water (10 msw) is also equal to 1 ATA. So if we dive to a depth of 10 m under the sea, we are subjected to a total pressure of 2 ATA. At a depth of 20 m sea water, the pressure is 3 ATA and so on.

Problems Faced by Marine Mammals During Deep Diving

What are the major problems for deep diving marine mammals? Firstly, the physical consequences of increased pressure on the animal's body. Secondly, the "bends", otherwise known as decompression sickness.

Physical Consequences of Increased Pressure

One of the most important effects imposed upon a marine mammal when it dives to depth is the increase in pressure on the spaces in the body containing air. The most important of these are the lungs and the main airway to the lungs, the *trachea.* To understand what can happen to these air-filled spaces when the animal dives, we must recall the relationship between gas pressure and volume.

If an inverted bucket with a volume of one litre containing air at the surface is forced to a depth of 10 msw, i.e., a pressure of 2 ATA, the volume of the air in the bucket will be halved, i.e., 0.5 litre. Thus, when the pressure is doubled, the volume is halved. At 20 msw the pressure increases three times and volume is now one-third of a litre and so on (Boyle's Law).

What happens to the lungs in a breath-hold diver? At the surface where the pressure is 1 ATA the lung volume is about six litres. At a depth of 10 msw the pressure is doubled and so the lung volume is halved to three litres. This decrease in lung volume is possible because the bony chest cage or thorax, which is composed of ribs and the breast-bone or sternum, can be compressed to some extent. But there is a depth limit at which compression of the chest wall can no longer occur and

Table 2. The maximum known depth and duration of dives for the same five species as shown in Table 1. (Adapted from Kooyman 1988 and Harrison and Kooyman 1971).

Species	Time min.	Depth m
Sperm Whale	75	1 140
Weddell Seal	70	600
Blue Whale	49	100
Bottle-nose Dolphin	12	535
Human (approx.)	2	10

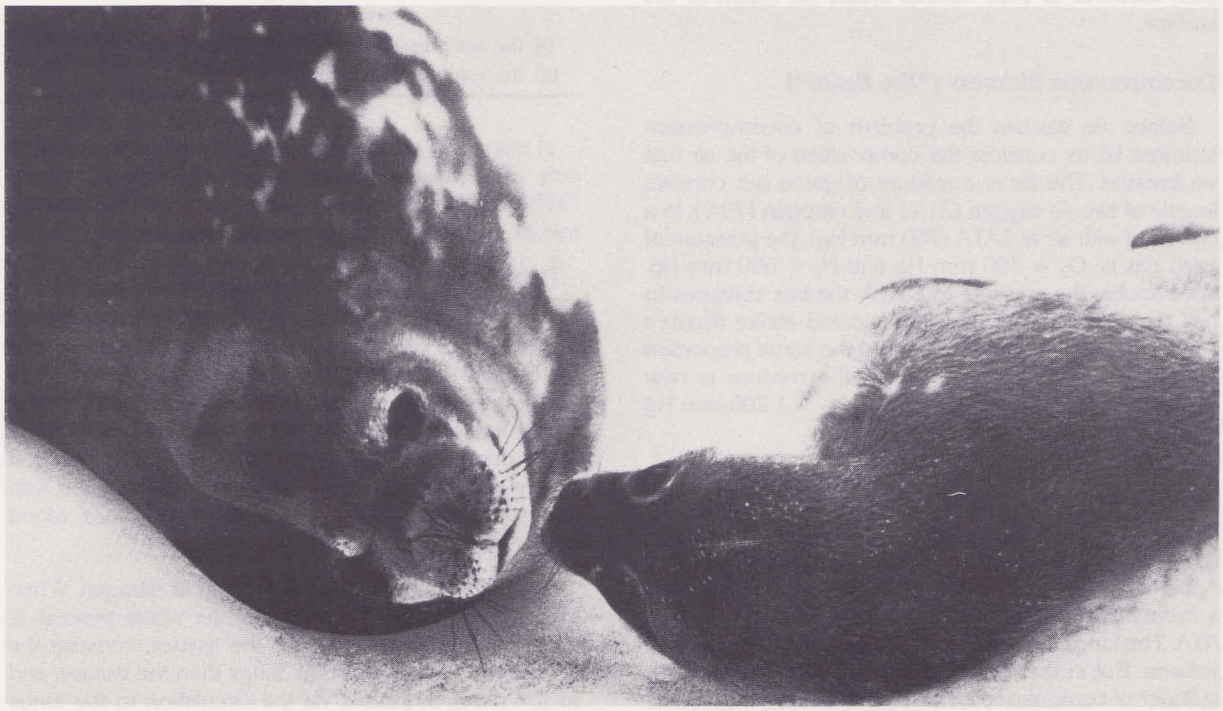

Figure 1. Mother and pup Weddell Seal at McMurdo Sound, Antarctica. Pups can double their birth weight in about 10 days. (Photograph kindly provided by Professor R. Elsner, Institute of Marine Science, University of Alaska, Fairbanks, Alaska).

any further increase in external water pressure would damage the chest wall and lung tissue. This traumatic collapse may happen at depths below 100 m and the condition in human divers is descriptively called "the squeeze".

How do Marine Mammals Avoid the Squeeze?

As we have seen, marine mammals can easily perform dives to depths five to ten times greater than a human being. How do they avoid the squeeze?

In many marine mammals the thorax is unusually flexible because of the looseness or lack of connection between the ribs and sternum. So the chest wall offers almost no resistance to compression and the thorax can collapse completely without damage no matter to what depth the seal dives. In addition the diaphragm is in an oblique position in relation to the spine and this arrangement also facilitates collapse of the thoracic cage and lungs (Harrison and Kooyman 1971).

In the human being the trachea is held open by horseshoe shaped cartilages. As the external water pressure rises the cartilages cannot collapse much without causing tissue damage. In the Weddell Seal the tracheal cartilages are shaped like a bow in cross section which allows them to be completely flattened out at depth and then rebound to their normal shape on return to the surface.

Decompression Sickness ("The Bends")

Before we discuss the problem of decompression sickness let us consider the composition of the air that we breathe. The air is a mixture of gases but consists largely of two — oxygen (21%) and nitrogen (79%). In a box filled with air at 1ATA (760 mm Hg), the pressure of each gas is: O_2 = 160 mm Hg and N_2 = 600 mm Hg. If we double the pressure to 2 ATA, the box collapses to half the volume as we have considered earlier (Boyle's Law). However, the gases are still in the *same proportion* but the pressure of each gas in the mixture is now doubled, i.e., O_2 = 320 mm Hg and N_2 = 1 200 mm Hg (Dalton's Law).

To examine the essential features of decompression sickness let us consider what happens to the human Scuba diver who breathes compressed air underwater (Scuba = Self-contained Underwater Breathing Apparatus). We have already discussed what happens to a *breath-hold* diver who descends to a depth of, say 4 ATA. The lungs are squeezed to one-quarter their surface volume. But in the case of a *Scuba diver* who carries a cylinder of compressed air on his/her back, the pressure of the air supplied to the diver's lungs via a face mask is automatically adjusted so that it is always at the same pressure as the water surrounding the body. This mechanism provides the diver with air to breath and prevents "the squeeze".

Now when a Scuba diver descends rapidly to a pressure of 4 ATA he/she will be breathing compressed air at 4 ATA. The gas pressure in the small air sacs, or *alveoli,* at the end of the lung airways will also be 4 ATA. But the pressure of the gas in the bloodstream passing through the lungs will still be near surface pressure of 1 ATA. So what happens?

Here we must note two basic factors that influence the amount of gas dissolved in a liquid (Table 3). The concentration of a gas in a liquid depends on the *gas pressure* above the liquid and the *solubility of the gas* in the liquid. So the greater the gas pressure, the more gas dissolves in the liquid and the greater the solubility of the gas in the liquid, the more gas dissolves in the liquid. The terms gas *pressure* and gas *tension* are used synonymously.

Table 3. Factors affecting the concentration of a gas dissolved in a liquid at a given temperature. (Adapted from Garside and Phillips 1957; Kooyman 1988).

$$Cg = Mg/V_L = Pg \times Sg$$

The concentration of gas, Cg, i.e., the number of molecules per unit volume of the liquid, Mg/V_L, at a given temperature is directly proportional to:

(i) the *gas pressure* above the liquid, Pg, and

(ii) the *solubility of the gas in the liquid,* Sg.

In respect of our discussion here, we are interested in only one constituent of the air gas mixture, namely NITROGEN, for it is the nitrogen which plays the critical role in decompression sickness.

Now let us return to a Scuba diver who has descended rapidly from the surface to depth.

First, let us consider the on-loading of nitrogen into the tissues. At the depth of 4 ATA the nitrogen diffuses from the lungs and dissolves in the blood stream which carries it out to tissues where it diffuses into the different tissue compartments. Some tissues have a good blood supply and these become filled up, i.e., *saturated* with nitrogen first. Conversely tissues with a poor blood supply become saturated last.

Now let us consider the off-loading of nitrogen. When a diver rises towards the surface the whole process is reversed. The nitrogen leaves the tissues, because the pressure is now lower in the lungs than the tissues, and so the nitrogen passes via the circulation to the lungs where it is exhaled. Tissues with good blood flow off-load fastest, those with poor blood flow are slowest.

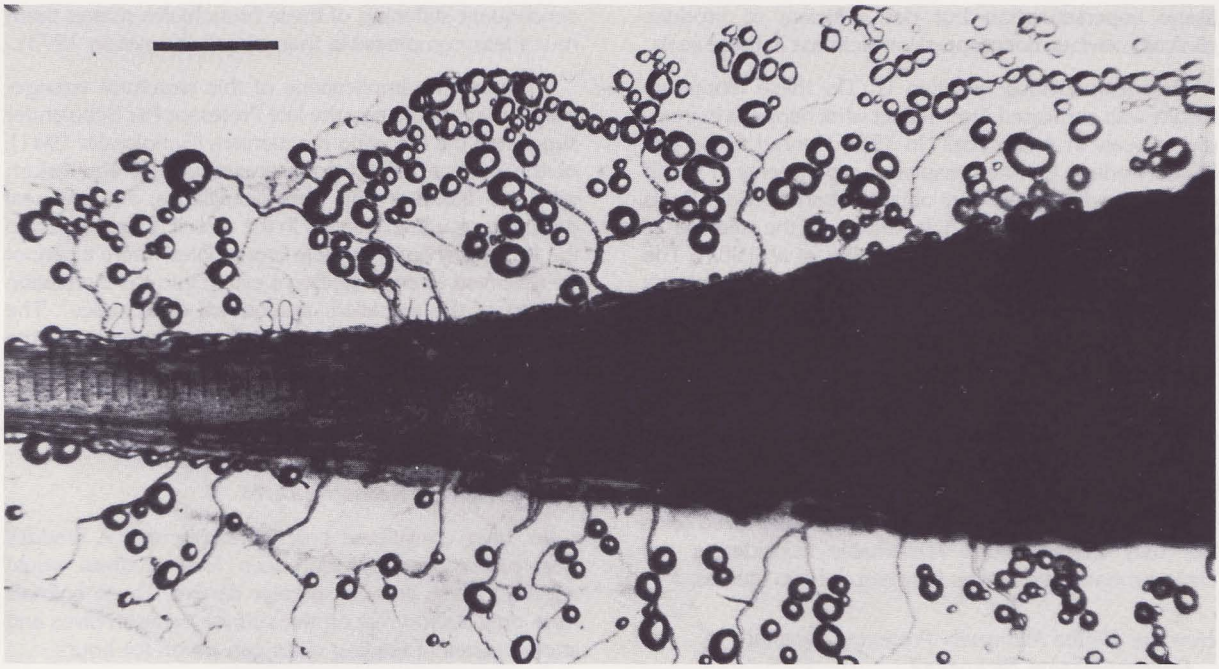

Figure 2. Strings of nitrogen bubbles in the blood vessels of a tadpole's tail after rapid decompression. Bar = 1 mm (From Gooden 1973. Illustration reproduced with kind permission of *Aust. J. Exp. Biol. Med. Sci.).*

But if a diver rises too rapidly to the surface, the nitrogen cannot be off-loaded from the body quickly enough to prevent the tissues and blood from developing an excess of nitrogen for that particular pressure. The tissues become *supersaturated* with nitrogen.

We have all seen what happens to dissolved gas when the pressure above the liquid is suddenly reduced when we open a bottle of soda water. The water fizzes with bubbles as gas, in this case carbon dioxide, comes out of solution. The same thing can happen to living tissue when the pressure around an animal is suddenly reduced from a high to a low pressure. Strings of bubbles develop inside blood vessels (Fig. 2). It is these bubbles of nitrogen that cause the clinical condition known as decompression sickness or "the bends". The major clinical features of decompression sickness may be classified under three headings depending upon whether the bubbles of nitrogen have formed in the musculoskeletal, nervous or lung tissue (Table 4).

Table 4. Major clinical features of decompression sickness.

1. The "bends" — pain in joints, muscles and bones	
2. Neurological effects —	Spinal cord → paralysis
	Brain → dizziness, deafness, loss of vision, loss of consciousness
3. The "chokes" — chest pain, shortness of breath, dry cough	

Does Decompression Sickness Occur in the Weddell Seal?

Overt decompression sickness in marine mammals appears to be extremely rare. I am aware of only one report of clinically obvious decompression sickness in a marine mammal (Scholander 1941).

In the early 1970s Dr Gerald Kooyman and his colleagues, working at Scripps Institution of Oceanography, subjected seals to *simulated* deep dives (Kooyman *et al.* 1972). The animals were placed in a pressure chamber in which they could be totally immersed. The pressure of the water-filled chamber could then be raised to the required level. Arterial blood samples were obtained before, during and after these simulated dives.

Simulated dive pressures for two Elephant Seals ranged from 4 ATA to 14.6 ATA. One minute after compression the nitrogen tension in the arterial blood rose sharply to 2.5 ATA. However, six minutes later the tension had fallen to about 1.5 ATA, presumably because the nitrogen was diffusing from the blood into body tissues. These blood tensions were up to eight times *lower* than the theoretical maximum nitrogen tension possible considering the pressures to which the seals were subjected. The decompression phase must have created some

tissue supersaturation but not sufficient to produce clinically obvious decompression sickness in these seals.

The next obvious question is "Do these laboratory results with simulated dives reflect what happens in freely diving seals in Antarctica?" In 1985 Konrad Falke and his co-workers reported results of experiments in which arterial blood samples were obtained from Weddell Seals diving freely through a hole drilled in the sea ice of McMurdo Sound in Antarctica (Falke *et al.* 1985). The equipment was attached to the seals under anaesthesia and after three to five days of monitoring, it was removed and the seals returned to their native colonies.

The average nitrogen tension during diving reached a maximum of 2.6 ATA at a depth of 70 m. There was no further increase in tension despite the fact that the seals descended below 200 m. On ascent the tension had actually decreased to 2.0 ATA. These levels were slightly higher than Kooyman's findings in laboratory dived seals but they corresponded remarkably considering the environmental differences between the two studies.

How are Marine Mammals Protected Against Decompression Sickness?

There are probably several mechanisms involved (Table 5).

Table 5. Summary. How marine mammals avoid decompression sickness.

1. *Limiting the nitrogen load*
(a) Reduction of lung volume at start of dive
(b) Shift of alveolar air into "dead space"
2. *Limiting nitrogen distribution*
Selective arterial constriction — the diving response
3. *Other possible factors*
(a) Efficient off-loading of surplus nitrogen
(b) Resistance to circulating bubbles

Limiting the Nitrogen Load

Seals usually dive with lungs only half full, so the amount of nitrogen taken to depth is much less compared with an animal diving with full lungs. In addition the amount of nitrogen that is absorbed from the lungs into the bloodstream could be limited. The gas exchange between the lungs and the blood flowing through them occurs at the tiny air pockets at the end of the airways — the alveoli. The structure of the small airways and alveoli of Sea Lion and Fur Seal lungs is quite unusual. The small airways, or *bronchioles,* are reinforced by unusually large amounts of cartilage and smooth muscle which extend down to the alveoli. The consequent stiffening of these bronchioles makes them much less compressible than alveoli (Kooyman 1973).

What are the implications of this structural arrangement? many years ago the late Professor Per Scholander suggested the following mechanism (Scholander 1941). At the surface the nitrogen pressure is in equilibrium with the blood in the lung capillaries. As the seal descends, much of the gas in the alveoli is squeezed into the less easily compressible bronchioles where it cannot be absorved to any significant extent into the circulation — part of the so-called "anatomical dead space". The nitrogen remaining in the alveoli is absorbed into the lung circulation. At even greater depths the residual gas in the alveoli is absorbed and the alveoli collapse completely (Kooyman *et al.* 1972).

The Problem of Repetitive Dives

We have considered only the single dive. A Weddell Seal performing repeated deep feeding dives would appear to face an even greater danger. These animals have only a short rest on the surface between dives and such a series of feeding dives can go on for hours!

Some years ago a Danish medical officer, Dr P. Paulev, developed decompression sickness after repeated breath-hold dives in a submariner's training tank (Paulev 1965). He later used a computer to demonstrate the danger of repeated two-minute dives with one minute on the surface between dives. With each dive a certain amount of nitrogen enters the body's tissues, but the one minute on the surface permits only a small portion of this gas to be off-loaded from the body. Thus dive by dive, the tissue nitrogen tension is "pumped up" until, in his case, on the sixteenth dive a critical level of nitrogen supersaturation occurred with resulting decompression sickness (Paulev 1967).

So what happens to tissue nitrogen when a marine mammal performs a series of deep dives?

To study this intriguing question Ridgway and Howard (1979) studied two navy trained dolphins who could perform a series of dives on command. The two Bottlenose Dolphins, named Brown and Blue, dived repeatedly for about one hour to a depth of 100 m. On each dive lasting about one-and-a-half minutes, they pressed a switch at 100 m with their snouts and returned to the surface to receive a food reward. They spent about one minute resting on the surface between each dive. Blue made 25 dives and Brown 23.

After its last dive each dolphin swam on to a platform which was brought alongside a larger craft. A small needle was inserted through the blubber layer into the skeletal muscle tissue and this was connected to a mass

spectrometer which could determine the tension of nitrogen in the muscle. The muscle nitrogen tension at the end of the series of dives could only be approximated since it took about eight minutes to position the animal and set up the measuring equipment. This estimate of muscle nitrogen tension was approximately 2 ATA, considerably less than the theoretical maximum level.

What Protects Marine Mammals from High Tissue Nitrogen Tension after Repeated *Deep Dives?*

Neither limiting the lung volume nor the nitrogen uptake from the lungs appears to play a significant role in dolphins. Some other factor or factors must be involved (Table 5).

Limitation of Nitrogen Distribution

The protection appears to result from the way the nitrogen is distributed by the circulation to the different body tissues. Here we must consider another fascinating phenomenon of breath-hold diving which can profoundly influence the distribution of blood flow and hence the distribution of nitrogen in the diving mammal (Fig. 3) (Elsner and Gooden 1983).

When the animal is breathing air on the surface the right side of the heart pumps blood through the lungs where it is oxygenated. The left side then pumps this oxygenated blood to the brain and to the other tissues of the body. Upon breath-hold diving there is marked constriction of arteries in a large proportion of the animal's body but not in the brain or heart circulation. This selective constriction of arteries markedly reduces or arrests blood flow to a large portion of the body tissue and hence conserves the oxygen for the heart and the brain which are particularly sensitive to oxygen lack and, of course, critical to the animal's survival.

Another outcome of such a restriction of the circulation in the deep-diving mammal is to confine the distribution of nitrogen to the heart-brain circuit and markedly reduce or prevent its distribution to tissues such as skeletal muscle and the fatty blubber layer.

The evidence discussed earlier suggests that there is significant restriction of blood flow to peripheral tissues, including skeletal muscle, in diving dolphins just as there is in seals. However, in some tissues complete arrest of flow may not be continuous throughout the dive. Blood flow may be pulsed in small volumes in supply arteries during diving to sustain oxygenation and therefore intermittent constriction of these arteries in tissues such as muscle (Gooden and Elsner 1985). Such a mechanism would explain the limited rise in muscle nitrogen tension observed in the diving dolphins.

In addition, given that a certain mass of nitrogen enters the circulation during diving, the smaller the circulating blood volume, the greater will be the nitrogen concentration and hence the nitrogen tension in the circulating blood (see equation in Table 3). It is therefore interesting to note that the arterial nitrogen tension was somewhat

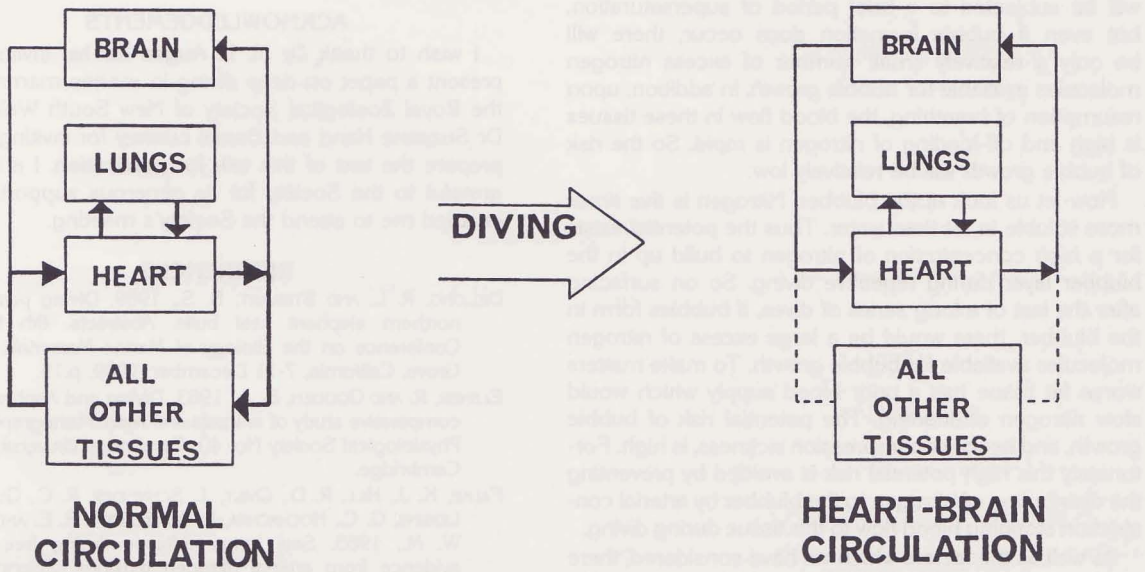

Figure 3. Schematic of the circulatory adjustments of the diving response. Left side — the normal circulation. Right side — circulation during diving. Dotted line represents arrest of blood flow to a large portion of body tissue but *not* including the heart-brain circuit. (Adapted from Gooden 1972.)

higher in freely diving Weddell Seals compared with seals subjected to simulated dives in the laboratory. This finding provides important evidence that selective vasoconstriction is at least as marked in freely diving seals as it is in laboratory based animals.

Physiological Priorities

We see here a possible conflict between physiological needs. On the one hand shut down of blood flow to non-critical tissues allows conservation of oxygen stores for the heart and brain and therefore longer underwater survival. On the other, distribution of the nitrogen load to greater circulating blood volume would result in a lower tissue nitrogen tension and, theoretically at least, a decreased risk of bubble formation and growth. Here is a fascinating conundrum.

Many questions arise. I have selected two.

Why do marine mammals not show signs of nitrogen bubbles in the brain and heart?

Why should flow be prevented in, say, the blubber layer?

To answer these questions let us consider the risk of bubble growth in the heart and brain tissue compared with the risk of bubble growth in the blubber (Table 6).

Firstly, let us look at the heart and brain. Both these tissues are high in water content and therefore have a relatively *low solubility* for nitrogen. Therefore, though the nitrogen *tension* in the arterial blood perfusing these tissues has been shown to be about 2 ATA, the *concentration* of nitrogen in these tissues will be relatively low.

On rising to the surface, the heart and brain tissues will be subjected to a brief period of supersaturation, but even if bubble formation does occur, there will be only a relatively small number of excess nitrogen molecules available for *bubble growth.* In addition, upon resumption of breathing, the blood flow in these tissues is high and off-loading of nitrogen is rapid. So the risk of bubble growth will be relatively low.

Now let us look at the blubber. Nitrogen is *five times* more soluble in fat than water. Thus the potential exists for a *high concentration* of nitrogen to build up in the blubber layer during repetitive diving. So on surfacing after the last of a long series of dives, if bubbles form in the blubber, there would be a large excess of nitrogen molecules available for bubble growth. To make matters worse fat tissue has a poor blood supply which would slow nitrogen off-loading. The potential risk of bubble growth, and hence decompression sickness, is high. Fortunately this high potential risk is avoided by preventing the distribution of nitrogen to the blubber by arterial constriction stopping blood flow to this tissue during diving.

As well as the factors which we have considered, there may well be others that play a part in protecting the marine mammals against decompression sickness. An increased rate of breathing and increased tissue blood flow above resting levels have the potential for increasing the rate of off-loading nitrogen. It seems quite likely that tiny bubbles may form in marine mammals performing repetitive dives but that they do not result in clinically observable effects. Are marine mammals more tolerant to circulating bubbles? Do the complex networks of veins found in these animals act as bubble traps? We now have the technology to start to answer some of these intriguing questions.

Table 6. Theoretical risk of bubble growth in heart-brain and blubber after repeated diving if no restriction of tissue blood flow.

Tissue compartment	Heart and brain (watery tissue)	Blubber (fatty tissue)
Nitrogen tension	Both 2 ATA	
Nitrogen solubility/ concentration	low	high
Excess N_2 molecules	few	many
Off-loading ability	excellent	poor
Risk of bubble growth	low	high

Challenges for the Future

Despite these fascinating studies, there is much we still do not understand. For example, we have hardly started to come to grips with the physiology of the massive Sperm and Blue Whales. Perhaps scientists of the future will have the facilities to cope with such problems. What secrets remain to be unlocked? Great adventures into the physiology of these marvellous animals still await enquiring minds of the future.

ACKNOWLEDGEMENTS

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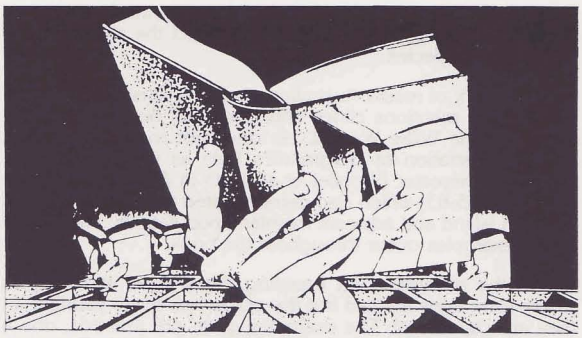
R.Z.S. Entomological Section

The Council has been informed that the Entomology Section is no longer in existence due to all members having transferred to some other group.

As of December 1989, therefore, there is NO official Entomological Section of the Royal Zoological Society of New South Wales, and therefore no publications or communications emanating from any such Section do not have the approval of the Society's Council.

If any member of the Society is interested in re-starting an entomological group within the Society's guidelines, Council would be pleased to hear from them.

Marianne Cochrane,
Hon. Secretary



BOOK REVIEWS

Ecology of Birds, An Australian Perspective

Hugh A. Ford

Published by Surrey Beatty & Sons Pty Ltd, Chipping Norton, NSW. Hardback, 230 × 150 mm, 287 pages with 48 colour plates, 39 black and white figures and 50 tables. Recommended retail price $36.90 plus postage.

This book is the latest in the Australian Ecology Series, originally published by University of Queensland Press. This is the second published by Surrey Beatty and Sons who have now taken over the series. In keeping with the other books from Surrey Beatty and Sons with which I am familiar, this book is very well produced and remarkably free from printing errors.

"Ecology of Birds" consists of 12 chapters which examine: the Australian environment and the origin of birds, distribution, food and foraging behaviour, community ecology, breeding, mating strategies and parental behaviour, co-operative breeding, population ecology, migration, the ecology of rare, endangered and extinct birds and concludes with a summary of the contributions that studies in the Australian region have made to understanding bird ecology and ecological theory as a whole. There is a brief section with a useful list of research priorities in the final few pages of the book.

Why was this book written and published? In the Foreword to *"Ecology of Birds"* Harold Heatwole, the General Editor of the series, writes "Regardless of emphasis, in each book of this series the available information in a particular field is reviewed critically and summarized so that the reader is brought abreast of current knowledge and developments." In the Preface, Hugh Ford notes "In this book I shall introduce a series of ecological topics from their current theoretical basis and then use such information as is available from the Australian region to examine these theories. Although I emphasize studies in Australia, I also use, where appropriate, work from New Zealand, New Guinea . . .". From these comments and part of the title "An Australian perspective", the reader should obtain a critical review of the current knowledge of avian ecology using up-to-date Australian or Australasian examples. However, the book does not provide a critical review of current knowledge, has some notable omissions and does not adequately emphasize Australian studies. I felt that the book is biased toward Northern Hemisphere studies and illustrations, even where there are